The following suggest that there may be a direct effect: and plaque rupture is well established. The participation of inflammatory cells and mediators in atherogenesis.

**INTRODUCTION:**
C-reactive protein: A person's baseline level of inflammation, as assessed by the plasma concentration of CRP predicts the long-term risk of a first MI [2], ischemic stroke, or peripheral artery disease [3]. Measurement of CRP levels improves risk stratification [4]. Several professional societies have issued statements or guidelines suggesting a role for the measurement of high sensitivity CRP in patients at intermediate risk for coronary heart disease, in whom measurement may help to direct further evaluation and therapy for primary prevention [5].

CRP is a member of the pentraxin family of proteins. It is an acute phase reactant synthesized mainly by the liver. Serum CRP levels are elevated in response to acute infections, inflammatory conditions and trauma. In these clinical situations, the serum CRP levels rise rapidly generally beyond 10 mg/L with a concomitant elevation of erythrocyte sedimentation rate (ESR). [6]

CRP has a relatively long half life of 18-20h, owing to its stable pentraxin structure. CRP levels do not exhibit diurnal variations in relation to food intake. High-sensitivity enzyme-linked immunosorbent assay (ELISA) and resonant acoustic profiling (RAP) can detect CRP with a sensitivity range of 0.01 to 10 mg/L. CRP is an acute phase protein that is produced predominantly by liver under the influence of cytokines such as interleukin (IL)-6 and tumor necrosis factor-alpha [1].

**BACKGROUND:** Inflammation is a known factor in the development of atherosclerosis and subsequent CVD events. Ongoing inflammation increases the vulnerability of an atherosclerotic lesion to erosion or rupture. The most extensively studied biomarker of inflammation is C-reactive protein (CRP) potentially linked to underlying atherosclerosis. For which standardized high-sensitivity assays (hs-CRP) are widely available which can detect CRP with a sensitivity range of 0.01 to 10 mg/L. CRP is an acute phase protein that is produced predominantly by liver under the influence of cytokines such as interleukin (IL)-6 and tumor necrosis factor-alpha [1].

**METHODS:** A study that aimed to analyze levels of hsCRP in determining morbidity and mortality in acute vascular events like Myocardial infarction (MI), unstable angina and stroke in 50 cases comparing to 50 controls with non vascular acute events. A total of 50 patients were enrolled for the study from the in-patient clinics of the Department of General Medicine and Department of cardiology. A single center matched case - control study design was chosen. Age and sex matched controls (50) were also studied for comparison and meaningful interpretation of data.

**RESULTS & CONCLUSION:** There is definite association of hsCRP in acute vascular events than other non vascular conditions, Very high prevalence of elevated hsCRP with acute vascular events. Level of hsCRP has no definite role in determining the mortality in patients with acute vascular events.

**KEYWORDS:**
HsCRP, Myocardial infarction, unstable angina, ischemic stroke

In addition to potentially promoting atherosclerosis, CRP may also have an adverse effect in myocardial infarction that may be mediated in part by an interaction with complement [11]. The value that constitutes an elevation in serum hs-CRP is not clearly defined. A statement from the Centers for Disease Control and Prevention and the American Heart Association (CDC/AHA) reached the following conclusions for the use of serum hs-CRP to estimate cardiovascular risk.

For the determination of cardiovascular risk, values were defined as, hs-CRP levels:
- $<1$ mg/L Low risk,
- 1 to 3 mg/L Intermediate risk,
- $>3$ mg/L High risk

It was suggested that a value above 10 mg/L should initiate a search for a source of infection or inflammation and the measurement of hs-CRP should be repeated in two weeks. Among apparently healthy men, the plasma concentration of CRP predicts the long-term risk of a first myocardial infarction, ischemic stroke, hypertension, peripheral vascular disease, sudden cardiac death, and total mortality.

**AIMS AND OBJECTIVES**
1. To determine the PREVALENCE OF ELEVATED HSCRP IN 50 PATIENTS WITH ACUTE VASCULAR EVENTS.
2. To compare with 50 controls of non vascular acute events.
3. To determine role of hsCRP in determining the morbidity and mortality.
4. To analyze the levels of hsCRP in patient without any other specific CVS risk factors.

**MATERIALS AND METHODS:**
**STUDY POPULATION**
A total of 50 patients were enrolled for the study from the inpatient wards of the Department of General Medicine and Department of cardiology.

Patients who are selected for the study satisfied all the inclusion and exclusion criteria. Written consent was obtained from all patients participating in the study.
Age and sex matched controls (50) were also studied for comparison and meaningful interpretation of data. The controls were recruited from other acute cases that were recruited from the wards; appropriate controls were recruited from the wards. All patients and controls were not from a single ethnic background.

**STUDY DURATION**
This study was conducted for a period of eighteen months from January 2015 to September 2016.

**STUDY DESIGN**
A single center matched case-control study design was chosen.

**METHODS**
Detailed clinical history was taken from each patient and a complete review of their case notes performed.
A complete clinical examination of the nervous system and cardiovascular system was done for each patient.

**LABORATORY METHODS**
To all selected patients, following investigations were taken.
- ECG
- ECHO
- CPK MB
- CT BRAIN
- FBS
- FLP
- hsCRP
- Other relevant investigations

HsCRP measured after admission in hospital within 1st 24hrs hsCRP were estimated by VITROS 5, 1 FS chemistry system and VITROS 5600 integrated system to quantitively measure CRP in human serum or plasma.

**INCLUSION CRITERIA FOR CASES**
1. Age more than 15 years.
2. Acute myocardial infarction [Evidenced by ECG, elevated CK MB, or ECHO]
3. Ischemic stroke [Two CT BRAIN taken at least 3 days gap showing signs of ischemia]
4. Unstable angina [Evidenced by ECG or elevated CK MB]

**FOR CONTROL**
1. Age more than 15 less than 60.
2. Presence of any illness
3. Event should be acute
4. No cardiovascular risk factors

**EXCLUSION CRITERIA**
1. Age less than 15 and more than 60
2. Smoking
3. Alcoholism
4. Patient with previous attacks
5. Other inflammatory conditions
   a. SLE
   b. Scleroderma
   c. Rheumatoid arthritis
   d. Other Connective Tissue Disorders
6. Immuno suppressant therapy
7. Patient last followup

**STATISTICAL ANALYSIS**
The significance of difference between two proportions was indicated by the chi-square test. Variables were considered to be significant if \( P < 0.05 \).

**RESULTS:**
In our study 50 patients were selected and 50 control were selected to analyze for the following:
To determine the PREVALENCE OF ELEVATED hsCRP in PATIENTS WITH ACUTE VASCULAR EVENTS.
- To compare with 50 controls of non vascular acute events.
- To determine role of hsCRP in determining the morbidity and mortality.
- To analyze the levels of hsCRP in patients without any other specific CVS risk factors

According to various references quoted above, the level of hsCRP determines morbidity as well as mortality.

When we divide and analyze as follows
1. With risk factors and elevated hsCRP.
2. Without risk factors only elevated hsCRP.
3. Controls

**CONTROLS:**

<table>
<thead>
<tr>
<th>NO OF PATIENTS</th>
<th>DISEASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>Fever</td>
</tr>
<tr>
<td>5</td>
<td>LRTI</td>
</tr>
<tr>
<td>3</td>
<td>Post operative</td>
</tr>
<tr>
<td>9</td>
<td>Trauma</td>
</tr>
<tr>
<td>3</td>
<td>Diabetic ketocadiois</td>
</tr>
<tr>
<td>3</td>
<td>Acute abdomen</td>
</tr>
<tr>
<td>9</td>
<td>pancreatitis</td>
</tr>
<tr>
<td>3</td>
<td>Uti</td>
</tr>
<tr>
<td>2</td>
<td>gastroenteritis</td>
</tr>
</tbody>
</table>

**SEX WISE DISTRIBUTION**

<table>
<thead>
<tr>
<th>GENDER</th>
<th>NO OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>MALE</td>
<td>34</td>
</tr>
<tr>
<td>FEMALE</td>
<td>16</td>
</tr>
</tbody>
</table>

**COMMUNITY WISE DISTRIBUTION**

<table>
<thead>
<tr>
<th>COMMUNITY</th>
<th>NO OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HINDU</td>
<td>43</td>
</tr>
<tr>
<td>MUSLIM</td>
<td>2</td>
</tr>
<tr>
<td>CHRISTIAN</td>
<td>5</td>
</tr>
</tbody>
</table>

**RISK FACTOR WISE DISTRIBUTION:**

<table>
<thead>
<tr>
<th>RISK FACTOR WISE DISTRIBUTION</th>
<th>TOTAL NO OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>HYPERTENSION</td>
<td>32</td>
</tr>
<tr>
<td>DIABETES</td>
<td>27</td>
</tr>
<tr>
<td>LDL&gt;100</td>
<td>25</td>
</tr>
<tr>
<td>HDL&lt;50 FOR FEMALE</td>
<td>20</td>
</tr>
<tr>
<td>&lt;40 FOR MALE</td>
<td>20</td>
</tr>
<tr>
<td>TGL &gt; 150</td>
<td>20</td>
</tr>
<tr>
<td>WITHOUT RISK FACTOR</td>
<td>11</td>
</tr>
</tbody>
</table>
In this study, it is found that:

The prevalence of elevated hs CRP in selected cases >0.3mg/L - 42/50 84%  
Prevalence of elevated hsCRP in control >0.3mg/L - 32/50 64%  

Prevalence of elevated hsCRP in Cases and Controls

<table>
<thead>
<tr>
<th>Type of Risk Factor</th>
<th>Low</th>
<th>Intermediate</th>
<th>High</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>HYPERTENSION</td>
<td>3</td>
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<td>3</td>
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<tr>
<td>DIABETES MELLITUS</td>
<td>3</td>
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<td>3</td>
<td>9</td>
</tr>
<tr>
<td>HYPERLIPIDEMIA</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>12</td>
</tr>
</tbody>
</table>

In considering the statistical significance, used Chi-square test and the result was obtained as follows:

According to Chi-square formula,  
\[ X^2 = 5.1975, \text{ Degree of freedom} = 1 \]  
The P value is 0.022619

While comparing for patients with risk factors and without risk factors, we have used Fisher exact test and the results are obtained as follows.

According to the above results, the P value is <0.05. So, this signifies that hsCRP has a definitive role in the cardiovascular pathology.

While analyzing the significance of mortality, we used the Fisher exact test and the results are obtained as follows.

Mortality in Cases with elevated hsCRP
In considering the statistical significance, used the fisher exact test and the result obtained is as follows: The two tailed P value is 0.5774.

The P value is >0.05. So this shows no statistical significance.

Therefore the level of hsCRP has no definite role in determining the mortality.

**DISCUSSION:**

In our study, 16 females were selected, one third of study population. Moreover, included 10% of minority ethics in our study and the study shows there is no significant variation with community. In our study there is no significant difference in morbidity and mortality between males and females.

In our study 35 persons have underlying cardiovascular risk factors. 18 patients have family history of cardiovascular events. 10 patients have no risk factors, but have elevated CRP.

The average elevated level of CRP in cases with risk factors and without risk factors are significantly above level of controls selected.

Regarding the distribution of cases without risk factors, seven cases are with intermediate risk value i.e., CRP between 1 and 3. Six cases are with low risk value.

Regarding the distribution of cases, thirty two cases are in intermediate risk value, fifteen cases are with low risk value and three patients are in high risk group.

Regarding the total mortality, most of the patients who died i.e. three in number belong to intermediate risk category, one person with low risk and one person with high risk.

On comparing the mortality in patients with various risk factors, hyperlipidemia plays a major role i.e. nearly four of the five deaths. Hypertension and diabetes are second most leading comorbid condition.

The study clearly shows hsCRP has a definite role in cardiovascular morbidity and mortality.

Studies with clinical CVD events included a case-control study by Rajeshwar et al [12] (1,156 subjects; hsCRP levels predict ischemic stroke), Goswami et al [13] (200 subjects; hsCRP is an independent predictor of CAD) and Guruprasad et al [14] (442 subjects; hsCRP levels are associated with an increasing severity of CAD).

A prospective cohort study by Rao et al [15] with 1,021 subjects, of whom 772 had established CAD and the rest were controls, found that hsCRP was an independent predictor of repeat coronary events.

**CONCLUSIONS AND SUMMARY**

In this study it is clearly shown that most of patient selected had:

- Very high prevalence of elevated hsCRP with acute vascular events.
- There is definite association of hsCRP in acute vascular events than other non vascular conditions.
- The analysis in patient without any specific cardiovascular risk factors shows definite relative correlations.
- Probably hsCRP may play a role in pathogenesis as mentioned in the literature above which needs further studies.
- Level of hsCRP has no definite role in determining the mortality in patients with acute vascular events.

**REFERENCES:**